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FILE COVERS 1907 - 12 Aug 2005  VOL 143 ISS 8
FILE LAST UPDATED: 11 Aug 2005  (20050811/ED)

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L3              STR
L4            2 SEA SSS SAM L3
L5           31 SEA SSS FUL L3

     FILE 'HCAPLUS' ENTERED AT 16:55:36 ON 12 AUG 2005
L6           21 SEA ABB=ON  PLU=ON  L5

     FILE 'HCAPLUS' ENTERED AT 16:55:56 ON 12 AUG 2005


     FILE HOME

     FILE REGISTRY
     Property values tagged with IC are from the ZIC/VINITI data file
     provided by InfoChem.

     STRUCTURE FILE UPDATES:   11 AUG 2005  HIGHEST RN 859751-76-1
     DICTIONARY FILE UPDATES:  11 AUG 2005  HIGHEST RN 859751-76-1

     New CAS Information Use Policies, enter HELP USAGETERMS for details.

     TSCA INFORMATION NOW CURRENT THROUGH JANUARY 18, 2005

       Please note that search-term pricing does apply when
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     ************************************************************
     *                                                          *
     * The CA roles and document type information have been removed from *
     * the IDE default display format and the ED field has been added,   *
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      * effective March 20, 2005.  A new display format, IDERL, is now    *
      * available and contains the CA role and document type information. *
      *                                                                   *
      *********************************************************************

      Structure search iteration limits have been increased.  See HELP SLIMITS
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      http://www.cas.org/ONLINE/DBSS/registryss.html

      FILE HCAPLUS

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      FILE COVERS 1907 - 12 Aug 2005  VOL 143 ISS 8
      FILE LAST UPDATED: 11 Aug 2005  (20050811/ED)

      New CAS Information Use Policies, enter HELP USAGETERMS for details.

       This file contains CAS Registry Numbers for easy and accurate
       substance identification.

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L3                STR
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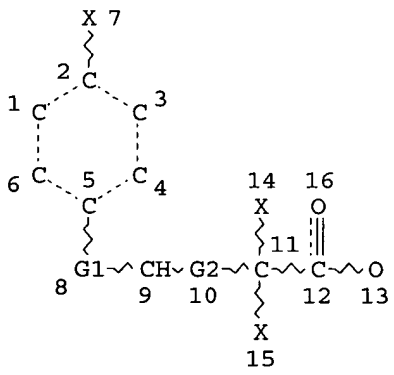


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NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
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RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 16

STEREO ATTRIBUTES: NONE
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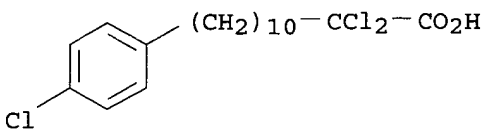
L6 ANSWER 1 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:548502 HCAPLUS
DOCUMENT NUMBER: 143:91301
TITLE: Skeletal muscle glycogen synthase subcellular localization: Effects of insulin and PPAR-α agonist (K-111) administration in rhesus monkeys
AUTHOR(S): Ortmeyer, Heidi K.; Adall, Yohannes; Marciani, Karina R.; Katsiaras, Andreas; Ryan, Alice S.; Bodkin, Noni L.; Hansen, Barbara C.
CORPORATE SOURCE: Obesity and Diabetes Research Center, Department of Physiology, School of Medicine, University of Maryland, Baltimore, MD, USA
SOURCE: American Journal of Physiology (2005), 288(6, Pt. 2), R1509-R1517
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Insulin covalently and allosterically regulates glycogen synthase (GS) and may also cause the translocation of GS from glycogen-poor to glycogen-rich locations. We examined the possible role of subcellular localization of GS and glycogen in insulin activation of GS in skeletal muscle of six obese monkeys and determined whether (1) insulin stimulation during a hyperinsulinemic euglycemic clamp and/or peroxisome proliferator-activated receptor (PPAR)-α agonist treatment (K-111, 3 mg/kg/day; Kowa) induced translocation of GS and (2) translocation of GS was associated with insulin activation of GS. GS and glycogen were present in all fractions obtained by differential centrifugation, except for the cytosolic fraction, under both basal and insulin-stimulated conditions. We found no evidence for translocation of GS by insulin. GS total (GST) activity was strongly associated with glycogen content (r = 0.70). Six weeks of treatment with K-111 increased GST activity in all fractions, except the cytosolic fraction, and mean GST activity, GS independent activity, and glycogen content were significantly higher in the insulin-stimulated samples compared with basal samples, effects not seen with vehicle. The increase in GST activity was strongly related to the increase in glycogen content during the hyperinsulinemic euglycemic clamp after K-111 administration (r = 0.74). Neither GS protein expression nor GS gene expression was affected by insulin or by K-111 treatment. We conclude that (1) in vivo insulin does not cause translocation of GS from a glycogen-poor to a glycogen-rich location in primate skeletal muscle and (2) the mechanism of action of K-111 to improve insulin sensitivity includes an increase in GST activity without an increase in GS gene or protein expression.
IT **221564-97-2**, K-111
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); BIOL (Biological study)
(insulin and PPARα agonist administration effect on skeletal muscle glycogen synthase subcellular localization in rhesus monkeys)
RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX

NAME)



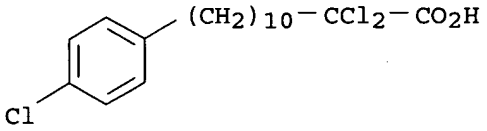
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:369898 HCAPLUS
DOCUMENT NUMBER: 143:946
TITLE: Effect of BM 17.0744, a PPARα ligand, on the metabolism of perfused hearts from control and diabetic mice
AUTHOR(S): Aasum, Ellen; Cooper, Marie; Severson, David L.; Larsen, Terje S.
CORPORATE SOURCE: Department of Medical Physiology, Institute of Medical Biology, Faculty of Medicine, University of Tromso, Tromso, N-9037, Norway
SOURCE: Canadian Journal of Physiology and Pharmacology (2005), 83(2), 183-190
CODEN: CJPPA3; ISSN: 0008-4212
PUBLISHER: National Research Council of Canada
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Peroxisome proliferator-activated receptor-α (PPARα) regulates the expression of fatty acid (FA) oxidation genes in liver and heart. Although PPARα ligands increased FA oxidation in cultured cardiomyocytes, the cardiac effects of chronic PPARα ligand administration in vivo have not been studied. Diabetic db/db mouse hearts exhibit characteristics of a diabetic cardiomyopathy, with altered metabolism and reduced contractile function. A testable hypothesis is that chronic administration of a PPARα agonist to db/db mice will normalize cardiac metabolism and improve contractile function. Therefore, a PPARα ligand (BM 17.0744) was administered orally to control and type 2 diabetic (db/db) mice (37.9±2.5 mg/(kg·d) for 8 wk), and effects on cardiac metabolism and contractile function were assessed. BM 17.0744 reduced plasma glucose in db/db mice, but no change was observed in control mice. FA oxidation was significantly reduced in BM 17.0744 treated db/db hearts with a corresponding increase in glycolysis and glucose oxidation; glucose and FA oxidation in control hearts was unchanged by BM 17.0744. PPARα treatment did not alter expression of PPARα target genes in either control or diabetic hearts. Therefore, metabolic alterations in hearts from PPARα-treated diabetic mice most likely reflect indirect mechanisms related to improvement in diabetic status in vivo. Despite normalization of cardiac metabolism, PPARα treatment did not improve cardiac function in diabetic hearts.

IT **221564-97-2**, BM 170744
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(effect of BM 17.0744, a PPARα ligand, on the metabolism of perfused hearts from control and diabetic mice)

RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:1124587 HCAPLUS
DOCUMENT NUMBER: 142:69188
TITLE: Combination therapy for the treatment of diabetes
INVENTOR(S): Erondu, Ngozi E.; Fong, Tung M.; MacNeil, Douglas J.; Van Der Ploeg, Leonardus H. T.; Kanatani, Akio
PATENT ASSIGNEE(S): Merck & Co., Inc., USA; Banyu Pharmaceutical Co., Ltd.
SOURCE: PCT Int. Appl., 109 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004110375	A2	20041223	WO 2004-US17291	20040602
WO 2004110375	A3	20050512		

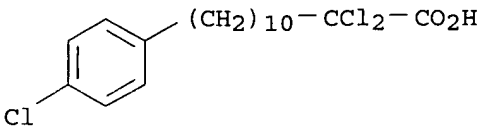
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-476388P P 20030606
OTHER SOURCE(S): MARPAT 142:69188

AB The present invention relates to compns. comprising an anti-obesity agent and an anti-diabetic agent useful for the treatment of diabetes, diabetes associated with obesity and diabetes-related disorders. The present invention further relates to methods of treating or preventing obesity, and obesity-related disorders, in a subject in need thereof by administering a composition of the present invention. The present invention further provides for pharmaceutical compns., medicaments, and kits useful in carrying out these methods.

IT **221564-97-2**, BM 170744
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination therapy of diabetes and diabetes-related disorders using antiobesity agent and antidiabetic agent and other agents)

RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



L6 ANSWER 4 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:1124581 HCAPLUS
DOCUMENT NUMBER: 142:69181
TITLE: Combination therapy for the treatment of hypertension
INVENTOR(S): Fong, Tung M.; Erondu, Ngozi E.; Macneil, Douglas J.; Mcintyre, James H.; Van Der Ploeg, Leonardus H. T.
PATENT ASSIGNEE(S): Merck & Co., Inc., USA
SOURCE: PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004110368	A2	20041223	WO 2004-US17090	20040602

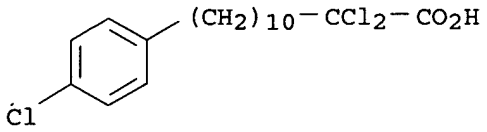
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-476390P P 20030606
OTHER SOURCE(S): MARPAT 142:69181

AB The present invention relates to compns. comprising an anti-obesity agent and an anti-hypertensive agent useful for the treatment of hypertension, hypertension associated with obesity, and hypertension-related disorders. The present invention further relates to methods of treating or preventing obesity, and obesity-related disorders, in a subject in need thereof by administering a composition of the present invention. The present invention further provides for pharmaceutical compns., medicaments, and kits useful in carrying out these methods.

IT **221564-97-2**, BM 170744
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination therapy of hypertension and hypertension-related disorders using antiobesity agent and antihypertensive agent and other agents and antihypertensive agent)

RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



L6 ANSWER 5 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:1037055 HCAPLUS
DOCUMENT NUMBER: 142:23019
TITLE: Preparation of 2,2-dichloro-12-(4-chlorophenyl)dodecanoic acid derivatives for treatment of diabetes and hyperlipemia
INVENTOR(S): Inoue, Keisuke; Toma, Tsutomu; Kitamura, Takahiro; Yamazaki, Yukiyoshi; Ishikawa, Tetsuya
PATENT ASSIGNEE(S): Kowa Company, Ltd., Japan
SOURCE: PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004103946	A1	20041202	WO 2004-JP7316	20040521
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RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005009909	A1	20050113	US 2004-849778	20040521
PRIORITY APPLN. INFO.:			US 2003-472737P P	20030523
			JP 2003-154372 A	20030530

OTHER SOURCE(S): MARPAT 142:23019
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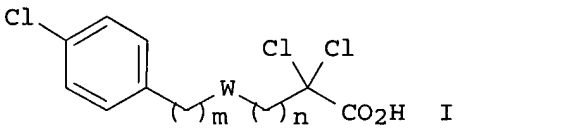

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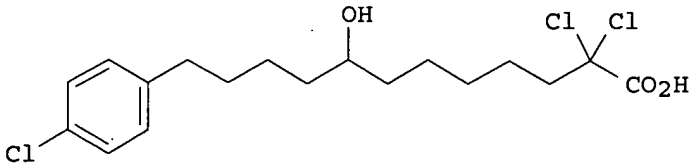

II

AB The title compds. I [wherein m = 0-4; n = 5-9; W = CH(OR); R = H, a protecting group, or CO], or salts or esters thereof are prepared For example, the compound II was prepared in a four-step synthesis. I showed efficiency in reduction of blood sugar, insulin, and triglyceride in rat. I are useful for the treatment for diabetes, complications of diabetes, hyperlipemia, etc. (no data).

IT **800395-14-6P 800395-16-8P 800395-18-0P**
800395-19-1P 800395-20-4P 800395-21-5P
800395-22-6P 800395-23-7P 800395-24-8P
800395-25-9P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(drug candidate; preparation of dodecanoic acid derivs. for treatment of diabetes and hyperlipemia)

RN 800395-14-6 HCAPLUS
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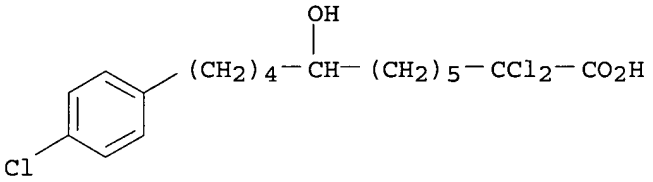

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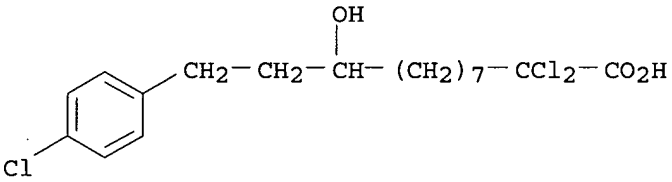

RN 800395-18-0 HCAPLUS
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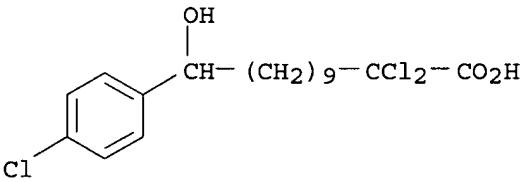

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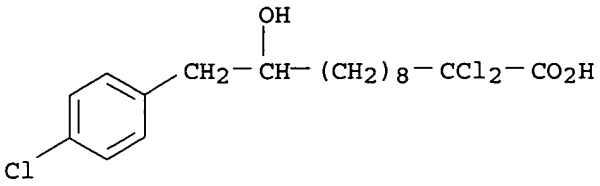
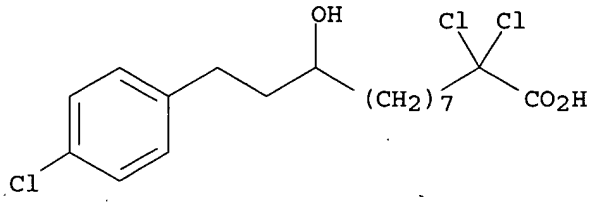


RN 800395-20-4 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-ι-hydroxy-, (+)- (9CI) (CA INDEX NAME)

Rotation (+).



RN 800395-21-5 HCAPLUS
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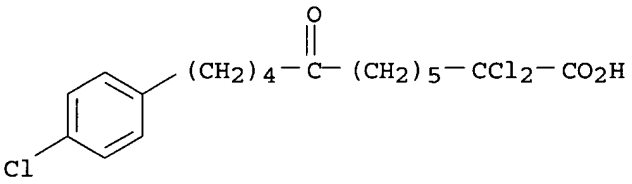

RN 800395-22-6 HCAPLUS
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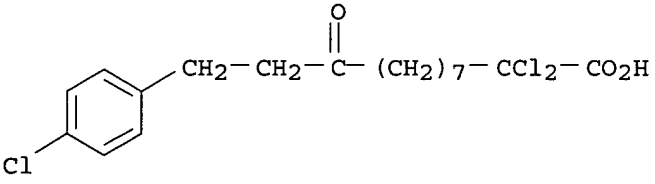
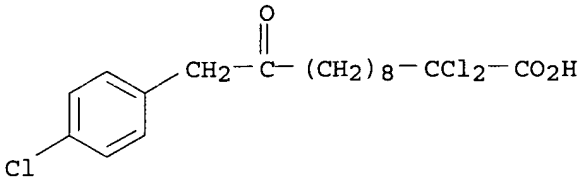


RN 800395-23-7 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-κ-oxo- (9CI) (CA INDEX NAME)



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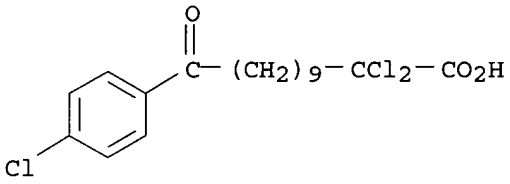
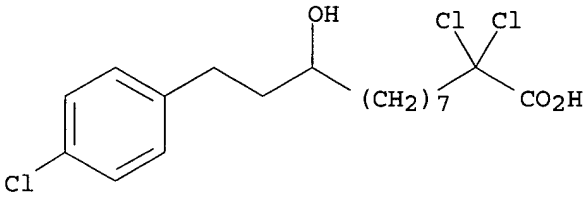


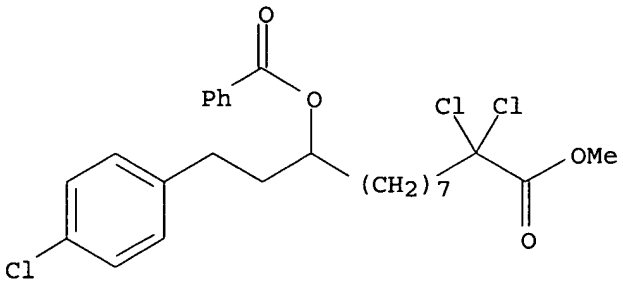
RN 800395-25-9 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-ι-hydroxy-, (-)- (9CI) (CA INDEX NAME)

Rotation (-).



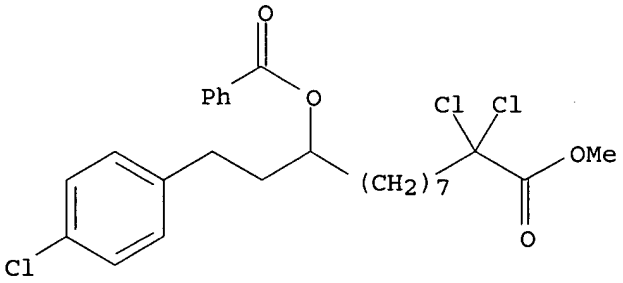
IT **800395-41-9P 800395-42-0P**
RL: PUR (Purification or recovery); RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(intermediate; preparation of dodecanoic acid derivs. for treatment of diabetes and hyperlipemia)
RN 800395-41-9 HCAPLUS
CN Benzenedodecanoic acid, ι-(benzoyloxy)-α,α,4-trichloro-, methyl ester, (+)- (9CI) (CA INDEX NAME)

Rotation (+).



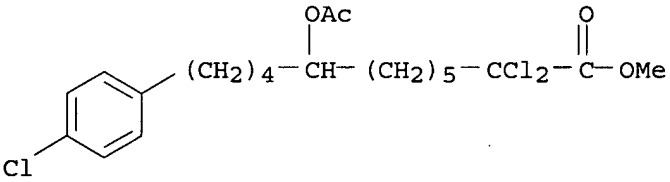
RN 800395-42-0 HCAPLUS
CN Benzenedodecanoic acid, ι-(benzoyloxy)-α,α,4-trichloro-, methyl ester, (-)- (9CI) (CA INDEX NAME)

Rotation (-).

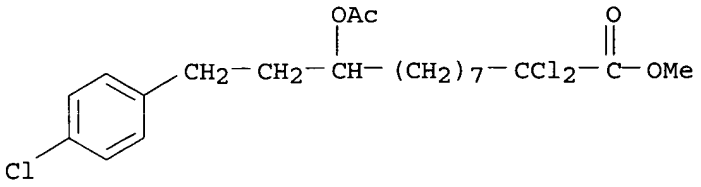


IT **800395-29-3P 800395-32-8P 800395-33-9P**
800395-34-0P 800395-35-1P 800395-36-2P
800395-38-4P 800395-40-8P 800395-44-2P
800395-45-3P 800395-46-4P 800395-48-6P
800395-49-7P 800395-50-0P 800395-51-1P
800395-52-2P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(intermediate; preparation of dodecanoic acid derivs. for treatment of diabetes and hyperlipemia)

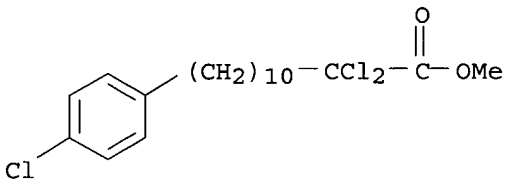
RN 800395-29-3 HCAPLUS
CN Benzenedodecanoic acid, η-(acetyloxy)-α,α,4-trichloro-, methyl ester (9CI) (CA INDEX NAME)



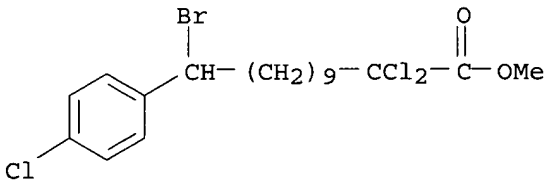
RN 800395-32-8 HCAPLUS
CN Benzenedodecanoic acid, ι-(acetyloxy)-α,α,4-trichloro-, methyl ester (9CI) (CA INDEX NAME)



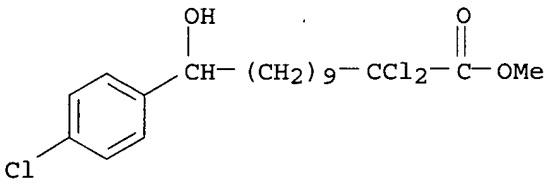
RN 800395-33-9 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-, methyl ester (9CI) (CA INDEX NAME)



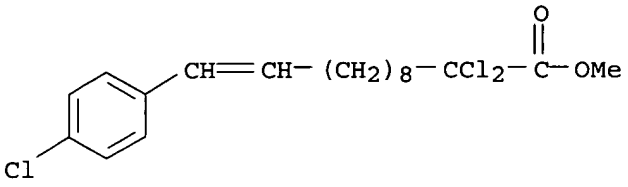
RN 800395-34-0 HCAPLUS
CN Benzenedodecanoic acid, λ-bromo-α,α,4-trichloro-, methyl ester (9CI) (CA INDEX NAME)



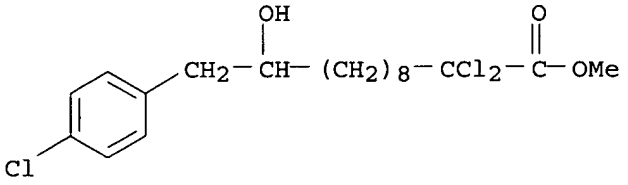
RN 800395-35-1 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-λ-hydroxy-, methyl ester (9CI) (CA INDEX NAME)



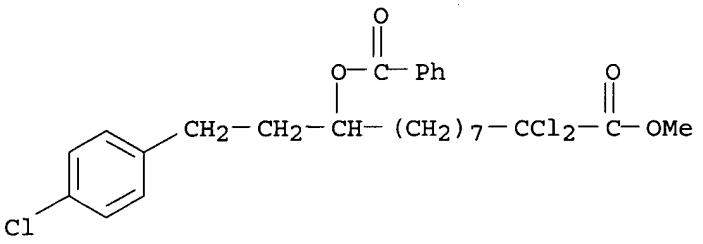
RN 800395-36-2 HCAPLUS
CN 11-Dodecenoic acid, 2,2-dichloro-12-(4-chlorophenyl)-, methyl ester (9CI) (CA INDEX NAME)



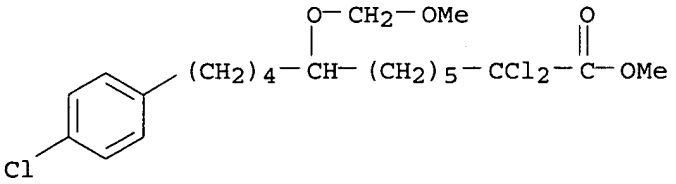
RN 800395-38-4 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-κ-hydroxy-, methyl ester (9CI) (CA INDEX NAME)



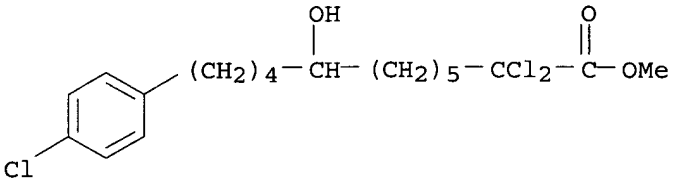
RN 800395-40-8 HCAPLUS
CN Benzenedodecanoic acid, ι-(benzoyloxy)-α,α,4-trichloro-, methyl ester (9CI) (CA INDEX NAME)



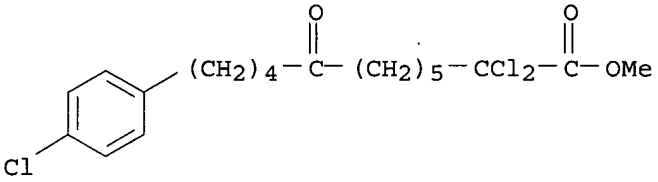
RN 800395-44-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-η-(methoxymethoxy)-, methyl ester (9CI) (CA INDEX NAME)



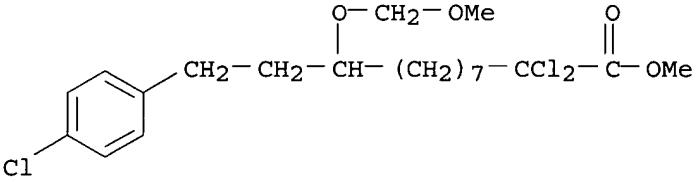
RN 800395-45-3 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-η-hydroxy-, methyl ester (9CI) (CA INDEX NAME)



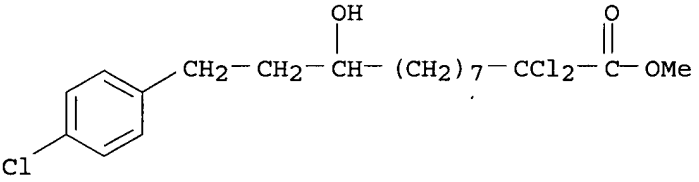
RN 800395-46-4 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-η-oxo-, methyl ester (9CI) (CA INDEX NAME)



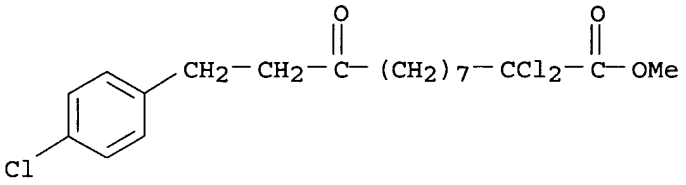
RN 800395-48-6 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-ι-(methoxymethoxy)-, methyl ester (9CI) (CA INDEX NAME)



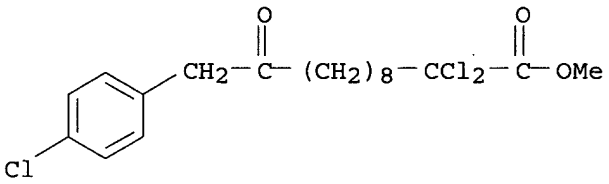
RN 800395-49-7 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-ι-hydroxy-, methyl ester (9CI) (CA INDEX NAME)



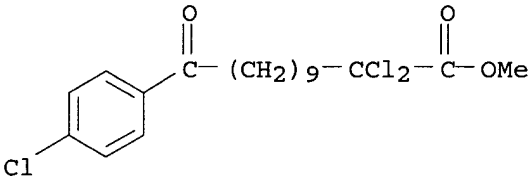
RN 800395-50-0 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-ι-oxo-, methyl ester (9CI) (CA INDEX NAME)



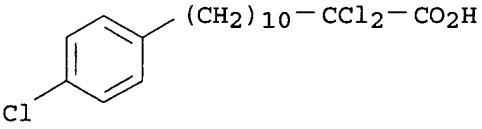
RN 800395-51-1 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-κ-oxo-, methyl ester (9CI) (CA INDEX NAME)



RN 800395-52-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-λ-oxo-, methyl ester (9CI) (CA INDEX NAME)



IT **221564-97-2**
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of dodecanoic acid derivs. for treatment of diabetes and hyperlipemia)
RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:906860 HCAPLUS
DOCUMENT NUMBER: 141:374603

TITLE: Human adipocyte fatty acid-binding protein (aP2) gene promoter-driven reporter assay discriminates nonlipogenic peroxisome proliferator-activated receptor γ ligands

AUTHOR(S): Rival, Yves; Stennevin, Aline; Puech, Laurence; Rouquette, Anne; Cathala, Claudie; Lestienne, Fabrice; Dupont-Passelaigue, Elisabeth; Patoiseau, Jean-Francois; Wurch, Thierry; Junquero, Didier

CORPORATE SOURCE: Centre de Recherche Pierre Fabre, Castres, Fr.

SOURCE: Journal of Pharmacology and Experimental Therapeutics (2004), 311(2), 467-475
CODEN: JPETAB; ISSN: 0022-3565

PUBLISHER: American Society for Pharmacology and Experimental Therapeutics

DOCUMENT TYPE: Journal

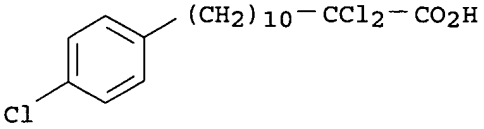
LANGUAGE: English

AB Peroxisome proliferator-activated receptors (PPARs) regulate storage and catabolism of fats and carbohydrates. PPARγ activity increases insulin sensitivity and adipocyte differentiation at the expense of adipogenesis and weight gain. The goal of this study was to (1) clone the promoter of the human adipocyte fatty acid binding protein (aP2) gene, namely fatty acid-binding protein-4, (2) characterize its pharmacol. regulation, and (3) determine its putative predictability for adipogenesis. Among the selected PPAR agonists, rosiglitazone and pioglitazone displayed the highest maximal efficacy (Emax) on reporter-gene assays in COS-7 cells cotransfected by either a galactosidase 4-response element-based or a human aP2 promoter-based Luc reporter vector, along with either chimeric or full-length human PPAR expression plasmids. The non-subtype-selective 2-(4-[2-(3-[2,4-difluorophenyl]-1-heptylureido)ethyl]phenoxy)-2-methyl-butyric acid (GW-2331) and the compds. [4-[3-(4-acetyl-3-hydroxy-2-propylphenoxy)-propoxyl]phenoxy]-acetic acid (L-165041), (4-((2S,5S)-5-(2-(bis(phenylmethyl)amino)-2-oxoethyl)-2-heptyl-4-oxo-3-thiazolidinyl)butyl)-benzoic acid (GW-0072), and indomethacin behaved as partial agonists relative to pioglitazone in full-length human aP2-PPARγ2. Beyond their partial PPARγ agonist properties, these compds. elicited a lower maximal up-regulation of mouse aP2 mRNA in 3T3-L1 adipocytes as compared with pioglitazone; these properties paralleled a time-dependent increase in neutral lipids. By contrast, the selective PPARα agonist 2,2-dichloro-12-(4-chlorophenyl)dodecanoic acid (BM-17.0744) neither stimulated the human aP2-PPARα promoter reporter-gene assay, thus demonstrating a specific interaction between PPARγ and the aP2 promoter, nor affected lipogenesis in 3T3-L1 cells. Altogether, these data characterized a functional promoter of the human aP2 gene; its in vitro pharmacol. regulation in PPARγ-mediated reporter-gene assay may represent an interesting complement or an alternative to time-consuming procedures aiming at discriminating PPAR ligands with low lipogenic properties.

IT **221564-97-2**, BM-170744
RL: PAC (Pharmacological activity); BIOL (Biological study)
(human adipocyte fatty acid-binding protein (aP2) gene promoter-driven reporter assay discriminates nonlipogenic peroxisome proliferator-activated receptor γ ligands)

RN 221564-97-2 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



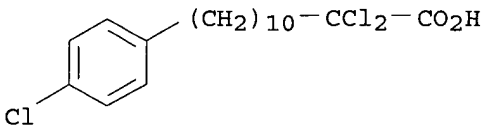
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:489210 HCAPLUS
DOCUMENT NUMBER: 141:184921
TITLE: Biochemical and morphological effects of K-111, a peroxisome proliferator-activated receptor (PPAR)α activator, in non-human primates
AUTHOR(S): Schafer, Silke A.; Hansen, Barbara C.; Volkl, Alfred; Fahimi, H. Dariush; Pill, Johannes
CORPORATE SOURCE: Institute of Anatomy and Cell Biology II, University of Heidelberg, Heidelberg, D-69120, Germany
SOURCE: Biochemical Pharmacology (2004), 68(2), 239-251
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB K-111 has been characterized as a potent peroxisome proliferator-activated receptor (PPAR)α activator. Antidiabetic potency and amelioration of disturbed lipid metabolism were demonstrated in rodents, which were accompanied by elevations of peroxisomal enzymes and liver weight To examine the possible therapeutic application of K-111 we have now assessed its efficacy in non-human primates with high transferability to humans. For this purpose obese, hypertriglyceridemic, hyperinsulinemic prediabetic rhesus monkeys were dosed sequentially with 0, 1, 3 and 10 mg/kg per day orally over a period of 4 wk each. In addition, the effect of K-111 on the peroxisome compartment was analyzed in cynomolgus monkeys using liver samples obtained following a 13-wk oral toxicity study. In prediabetic monkeys, the reduction of hyperinsulinemia and improvement of insulin-stimulated glucose uptake rate indicated amelioration of insulin resistance. These effects were nearly maximal at a dose of 3 mg/kg per day, while triglycerides and body weight were lowered significantly in a dose-dependent manner. This reduction of body weight contrasts sharply with the adipogenic response observed with thiazolidinediones, another family of insulin-sensitizing agents. In young cynomolgus monkeys at a dosage of 5 mg/kg per day and more, K-111 induced an up to three-fold increase in lipid β-oxidation enzymes with an 1.5- to 2-fold increase in peroxisome volume d. This moderate increase in peroxisomal activity by K-111 in monkeys is consistent with its role as an PPARα activator and corresponds to the observations with fibrates in other low responder mammalian species. The increase in β-oxidation may explain, at least in part, the lipid modulating effect as well as the antidiabetic potency of K-111. This pharmacol. profile makes K-111 a highly promising drug candidate for clin. applications in the treatment of type 2 diabetes, dyslipidemia, obesity and the metabolic syndrome.

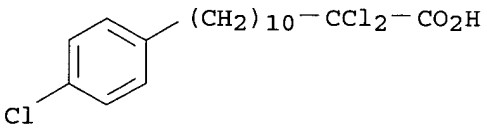
IT **221564-97-2**, K111
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(biochem. and morphol. effects of PPARα activator K-111 in non-human primates)

RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:388122 HCAPLUS
DOCUMENT NUMBER: 142:32750
TITLE: The effects of K-111, a new insulin-sensitizer, on metabolic syndrome in obese prediabetic rhesus monkeys. [Erratum to document cited in CA140:246684]
AUTHOR(S): Im, Wonpil; Feig, Michael; Brooks, Charles L., III
CORPORATE SOURCE: Department of Molecular Biology and Center for Theroetical Biological Physics, The Scripps Research Institute, La Jolla, CA, USA
SOURCE: Biophysical Journal (2004), 86(5), 3330
CODEN: BIOJAU; ISSN: 0006-3495
PUBLISHER: Biophysical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The corrected version of Figure 6 is given.
IT **221564-97-2**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(K 111; effects of K-111 as insulin sensitizer on metabolic syndrome in obese prediabetic rhesus monkeys (Erratum))
RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



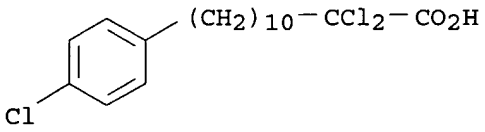
L6 ANSWER 9 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:917338 HCAPLUS
DOCUMENT NUMBER: 140:246684
TITLE: The effects of K-111, a new insulin-sensitizer, on metabolic syndrome in obese prediabetic rhesus monkeys
AUTHOR(S): Bodkin, N. L.; Pill, J.; Meyer, K.; Hansen, B. C.
CORPORATE SOURCE: Obesity and Diabetes Research Center, School of Medicine, Dept. of Physiology, University of Maryland, Baltimore, MD, USA
SOURCE: Hormone and Metabolic Research (2003), 35(10), 617-624
CODEN: HMMRA2; ISSN: 0018-5043

PUBLISHER: Georg Thieme Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

AB K-111, formerly BM 17.0744, (2,2-dichloro-12-(4-chlorophenyl)-dodecanoic acid) is a new insulin-sensitizer with peroxisome proliferator-activated receptor (PPAR) alpha activity but without PPAR gamma activity. We determined the efficacy of K-111 in non-human primates in increasing insulin-stimulated glucose uptake and improving metabolic syndrome, assessing the general health-related effects. Six adult male obese normoglycemic prediabetic and insulin-resistant rhesus monkeys were studied on vehicle and following K-111 treatment (four-week chronic dosing each of 3 doses: 1, 3, and 10 mg/kg/d) with assessment of changes in substrate, hormone, and blood pressure measurements and alterations in insulin sensitivity using the euglycemic, hyperinsulinemic clamp technique. K-111 led to significantly decreased body weight and improved hyperinsulinemia, insulin sensitivity, hypertriglyceridemia, and HDL-cholesterol levels without adipogenesis or significant effects on fasting glucose, 24-h urine glucose excretion, systolic or diastolic blood pressure, plasma fibrinogen, total cholesterol, or chemical and hematol. profile. These benefits are similar to the health-improving effects of calorie restriction, providing preliminary evidence that K-111 has excellent potential as a calorie-restriction mimetic agent. These results indicate the necessity of future study of K-111 for metabolic syndrome in humans, and suggest potential in reducing the risks of diabetes and cardiovascular disease.

IT **221564-97-2**, K 111 (Pharmaceutical)
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(K 111; effects of K-111, a new insulin-sensitizer, on metabolic syndrome in obese prediabetic rhesus monkeys)

RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:737571 HCAPLUS
DOCUMENT NUMBER: 139:255357
TITLE: Use of PPAR alpha agonists for the treatment of vascular and renal diseases
INVENTOR(S): Zahradka, Peter; Taylor, Carla
PATENT ASSIGNEE(S): Can.
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

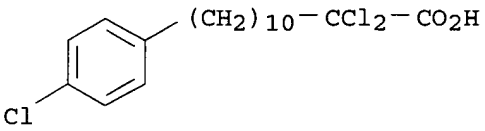
WO 2003075911	A1	20030918	WO 2003-CA335	20030311
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2481371	AA	20030918	CA 2003-2481371	20030311
PRIORITY APPLN. INFO.:			US 2002-362243P P	20020311
			WO 2003-CA335 W	20030311

AB Activation of peroxisome proliferator activated receptor alpha (PPARα) by administration of therapeutic amts. of a PPARα agonist, WY-14643, inhibits the proliferation of vascular smooth muscle cells, hepatoma cells and human renal proximal tubule cells. WY-14643 may be applicable as a medicament for the treatment of proliferative vascular disease (atherosclerosis, hypertension), revascularization-induced injury (restenosis) and chronic renal failure.

IT **221564-97-2**, BM 170744
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(PPARα agonist; PPARα agonists for treatment of vascular and renal diseases)

RN 221564-97-2 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 11 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER:	2003:570806 HCAPLUS
DOCUMENT NUMBER:	139:106503
TITLE:	Medicinal composition containing 2,2-dichloro-12-(4-chlorophenyl)dodecanoic acid
INVENTOR(S):	Kobayashi, Shinichiro; Takano, Niichiro; Kawashima, Hiroyuki; Shinoda, Yasuo; Inagi, Toshio
PATENT ASSIGNEE(S):	Kowa Co., Ltd., Japan
SOURCE:	PCT Int. Appl., 13 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	Japanese
FAMILY ACC. NUM. COUNT:	1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003059337	A1	20030724	WO 2003-JP251	20030115

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2474014	AA	20030724	CA 2003-2474014	20030115
BR 2003007172	A	20041103	BR 2003-7172	20030115
EP 1473033	A1	20041103	EP 2003-700558	20030115

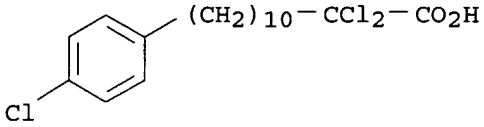
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

PRIORITY APPLN. INFO.:	JP 2002-7022	A	20020116
	WO 2003-JP251	W	20030115

AB Disclosed is a medicinal composition with excellent stability which contains a mixture of croscarmellose sodium and a substance selected from the group consisting of 2,2-dichloro-12-(4-chlorophenyl)dodecanoic acid (I), salts thereof, and esters thereof. For example, a tablet was formulated containing I·Na salt 1, croscarmellose sodium 12, lactose 115, hydroxypropyl cellulose 4.8, and Mg stearate 1.2 mg. After 2 wk storage at 60°, tablets contained 93.7 % of I, as compared to 84.9 % in control tablets containing hydroxypropyl cellulose instead of croscarmellose sodium.

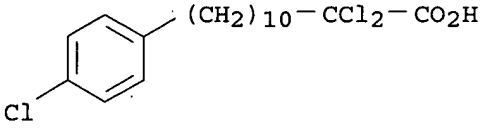
IT **178671-17-5 221564-97-2**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(stable formulations of 2,2-dichloro-12-(4-chlorophenyl)dodecanoate)

RN 178671-17-5 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro-, sodium salt (9CI) (CA INDEX NAME)



● Na

RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 12 OF 21 HCAPLUS

ACCESSION NUMBER: 2003:570770 HCAPLUS
DOCUMENT NUMBER: 139:111710
TITLE: Combinations of peroxisome proliferator-activated receptor-α agonists and cyclooxygenase-2 selective inhibitors, and therapeutic uses therefor
INVENTOR(S): Obukowicz, Mark G.
PATENT ASSIGNEE(S): Pharmacia Corporation, USA
SOURCE: PCT Int. Appl., 155 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

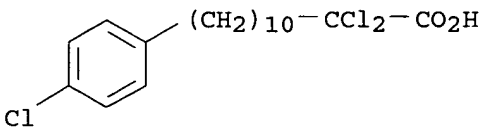
PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2003059294	A2	20030724	WO 2003-US956		20030114
WO 2003059294	A3	20050714			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
US 2003212138	A1	20031113	US 2003-341217		20030113
CA 2472168	AA	20030724	CA 2003-2472168		20030114
PRIORITY APPLN. INFO.:			US 2002-348297P	P	20020114
			US 2003-341217	A	20030113
			WO 2003-US956	W	20030114

OTHER SOURCE(S): MARPAT 139:111710

AB Methods for the treatment, prevention, or inhibition of pain, inflammation, or an inflammation-related disorder, and for the treatment or inhibition of a cardiovascular disease or disorder, and for the treatment or inhibition of cancer, and for the treatment of Alzheimer's disease in a subject in need of such treatment, prevention, or inhibition, include treating the subject with a peroxisome proliferator activated receptor-α agonist and a cyclooxygenase-2 selective inhibitor (e.g. celecoxib; preparation described), or prodrug thereof. Compns., pharmaceutical compns., and kits for effecting the particular methods are also described.

IT **221564-97-2**, BM170744
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(PPAR-α agonist combination with cyclooxygenase-2 selective inhibitor, and therapeutic use)

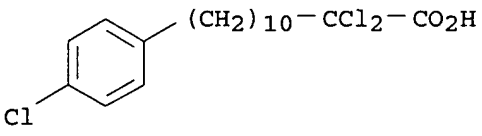
RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



L6 ANSWER 13 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:112419 HCAPLUS
DOCUMENT NUMBER: 138:301889
TITLE: Age-dependent changes in metabolism, contractile function, and ischemic sensitivity in hearts from db/db mice
AUTHOR(S): Aasum, Ellen; Hafstad, Anne D.; Severson, David L.; Larsen, Terje S.
CORPORATE SOURCE: Department of Medical Physiology, Faculty of Medicine, University of Tromsoe, Tromsoe, N-9037, Norway
SOURCE: Diabetes (2003), 52(2), 434-441
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Glucose and palmitate metabolism and contractile function were measured with ex vivo perfused working hearts from control (db/+) and diabetic (db/db) female mice at 6, 10-12, and 16-18 wk of age. Palmitate oxidation was increased by 2.2-fold in 6-wk-old db/db hearts and remained elevated in 10- to 12- and 16- to 18-wk-old hearts. Carbohydrate oxidation was normal at 6 wk but was reduced to 27 and 23% of control at 10-12 and 16-18 wk, resp. At 6 wk, db/db hearts exhibited a slight reduction in mech. function, whereas marked signs of dysfunction were evident at 10-12 and 16-18 wk. Mech. function after ischemia-reperfusion was examined in hearts from male mice; at 6 wk, db/db hearts showed normal recovery, whereas at 12 wk it was markedly reduced. Fatty acid oxidation was the predominant substrate used after reperfusion. Thus, diabetic db/db hearts exhibit signs of a progressive cardiomyopathy; increased fatty acid oxidation preceded redns. in carbohydrate oxidation Postischemic recovery of function was reduced in db/db hearts, in parallel with age-dependent changes in normoxic contractile performance. Finally, peroxisome proliferator-activated receptor-α treatment (3 wk) did not affect sensitivity to ischemia-reperfusion, even though carbohydrate oxidation was increased and palmitate oxidation was decreased.

IT **221564-97-2**, BM170744
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(BM170744 effect on changes in glucose and palmitate metabolism, contractile function, and ischemic sensitivity in hearts from db/db mice)
RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 14 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:717367 HCAPLUS
DOCUMENT NUMBER: 138:265466
TITLE: Cardiac function and metabolism in Type 2 diabetic

mice after treatment with BM 17.0744, a novel PPAR-α activator

AUTHOR(S): Aasum, Ellen; Belke, Darrell D.; Severson, David L.; Riemersma, Rudolph A.; Cooper, Marie; Andreassen, Morten; Larsen, Terje S.

CORPORATE SOURCE: Department of Medical Physiology, University of Tromso, Tromso, N-9037, Norway

SOURCE: American Journal of Physiology (2002), 283(3, Pt. 2), H949-H957
CODEN: AJPHAP; ISSN: 0002-9513

PUBLISHER: American Physiological Society

DOCUMENT TYPE: Journal

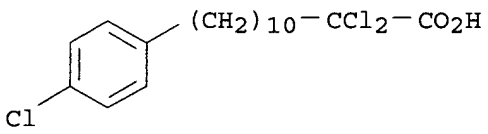
LANGUAGE: English

AB Hearts from diabetic db/db mice, a model of Type 2 diabetes, exhibit left ventricular failure and altered metabolism of exogenous substrates. Peroxisome proliferator-activated receptor-α (PPAR-α) ligands reduce blood plasma lipid and glucose concns. and improve insulin sensitivity in db/db mice. Consequently, the effect of 4- to 5-wk treatment of db/db mice with a novel PPAR-α ligand (BM 17.0744; 25-38 mg·kg-1·day-1), commencing at 8 wk of age, on ex vivo cardiac function and metabolism was determined Elevated plasma concns. of glucose, fatty acids, and triacylglycerol (34.0, 2.0, and 0.9 mM, resp.) were reduced to normal after treatment with BM 17.0744 (10.8, 1.1, and 0.6 mM). Plasma insulin was also reduced significantly in treated compared with untreated db/db mice. Chronic treatment of db/db mice with the PPAR-α agonist resulted in a 50% reduction in rates of fatty acid oxidation, with a concomitant increase in glycolysis (1.7-fold) and glucose oxidation (2.3-fold). Correction of the diabetes-induced abnormalities in systemic and cardiac metabolism after BM 17.0744 treatment did not, however, improve left ventricular contractile function.

IT **221564-97-2**, BM170744
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cardiac function and metabolism in type 2 diabetic mice after treatment with BM 17.0744)

RN 221564-97-2 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 15 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:56491 HCAPLUS

DOCUMENT NUMBER: 137:73203

TITLE: Pharmacological analysis of wild-type α, γ and δ subtypes of the human peroxisome proliferator-activated receptor

AUTHOR(S): Wurch, T.; Junquero, D.; Delhon, A.; Pauwels, P. J.

CORPORATE SOURCE: Department of Cellular and Molecular Biology, Centre de Recherche Pierre Fabre, Castres, 81106, Fr.

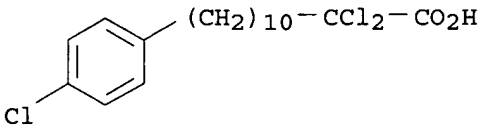
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology (2002), 365(2), 133-140
CODEN: NSAPCC; ISSN: 0028-1298
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Three distinct peroxisome proliferator-activated receptor (PPAR) cDNAs were isolated from human brain RNA. Whereas the PPARδ subtype perfectly matched the amino acid sequences reported in the Genbank database, several differences were found for the PPARα (Lys123Met, Ala268Val, Gly296Ala and Val444Ala) and PPARγ2 (Met8Ile, Pro9Ala, Met186Ile, Pro187Ala and the deletion of a Gln213 residue) subtypes. A pharmacol. anal. was undertaken by co-expressing each PPAR subtype with a reporter plasmid containing a luciferase gene under the transcriptional control of a synthetic, triplicated PPAR response element in either HepG2 or Cos-7 cells. Whereas fenofibrate unselectively activated the PPARα and PPARδ subtypes, the related BM-17.0744 compound was more potent and selective for PPARα. The thiazolidine dione derivs. rosiglitazone and pioglitazone were potent and selective PPARγ2 agonists. L-165041, reported as a selective and potent PPARδ ligand, displayed in this specified transactivation system, apart from its highly efficacious PPARδ agonist activity, partial and full agonism at, resp., PPARα and PPARγ2 subtypes. In conclusion, transcriptional control of a luciferase gene by wild-type PPAR subtypes provides powerful recombinant assays to evaluate ligand's efficacy at these nuclear receptors.

IT **221564-97-2**
RL: PAC (Pharmacological activity); BIOL (Biological study)
(pharmacol. anal. of wild-type α, γ and δ subtypes of human peroxisome proliferator-activated receptor)

RN 221564-97-2 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 16 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:604643 HCAPLUS
DOCUMENT NUMBER: 135:366677

TITLE: Peroxisome proliferator-activated receptor-α ligands inhibit cardiac lipoprotein lipase activity

AUTHOR(S): Carroll, Rogayah; Severson, David L.
CORPORATE SOURCE: Department of Pharmacology and Therapeutics, University of Calgary, Calgary, AB, T2N 4N1, Can.

SOURCE: American Journal of Physiology (2001), 281(2, Pt. 2), H888-H894
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

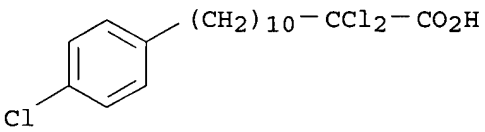
AB Peroxisome proliferator-activated receptors (PPARs) are ligand-activated

transcription factors that regulate gene expression of lipoprotein lipase (LPL) in liver and adipose tissue. We examined the direct effect of PPAR-α ligands on LPL catalytic activity in cultured cardiomyocytes from adult rat heart. After overnight culture (16 h), 1 μM Wy-14643 and 10 μM BM-17.0744 decreased total cellular LPL activity to .apprx.50% of control with no change in enzyme synthesis or mass; as a consequence, PPAR-α activation produced a significant decrease in LPL specific activity (mU/ng LPL protein). Wy-14643 and BM-17.0744 also reduced heparin-releasable LPL activity and mass in the culture medium. Inhibition of LPL activity by Wy-14643 did not reduce the ability of insulin plus dexamethasone to stimulate cellular and heparin-releasable LPL activities. A similar inhibitory effect on cellular and heparin-releasable LPL activity was observed when cardiomyocytes were cultured with 60 μM linoleic acid. In conclusion, two different PPAR-α ligands (Wy-14643 and BM-17.0744) inhibited cellular LPL activity in cultured cardiomyocytes by a posttranscriptional and posttranslational mechanism.

IT **221564-97-2**, BM-170744
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(peroxisome proliferator-activated receptor-α ligands inhibit cardiac lipoprotein lipase activity)

RN 221564-97-2 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 17 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:86550 HCAPLUS
DOCUMENT NUMBER: 132:216399
TITLE: Reduction of risk factors for cardiovascular complications by BM 17.0744
AUTHOR(S): Pill, Johannes; Meyer, Kirstin
CORPORATE SOURCE: Therapeutics Research, Roche Diagnostics GmbH, Mannheim, Germany
SOURCE: Cardiovascular Drug Reviews (1999), 17(3), 246-264
CODEN: CDREEA; ISSN: 0897-5957
PUBLISHER: Neva Press
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

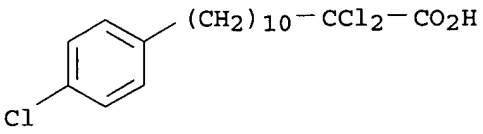
AB A review with 51 refs. is given on 2,2-dichloro-12-(p-chlorophenyl)dodecanoic acid (BM 17.0744). BM 17.0744 is a structurally new insulin sensitizer with intense antihyperglycemic and antihyperinsulinemic potency. Its mechanism of action involves lowering of triglycerides and free fatty acids mediated by induction of catabolic enzymes at a nuclear level. Insulin resistance, the key factor in the pathophysiol. of type 2 diabetes, is ameliorated in diabetic and prediabetic states. A toxicol. profile similar to that known from fibrates is expected. The potential risks with BM 17.0744 should be viewed in comparison with the structurally unrelated insulin sensitizers

of the thiazolidinedione (TZD) family. BM 17.0744 has a broad-spectrum effect on disturbed metabolism and is expected to reduce the incidence of cardiovascular diseases.

IT **221564-97-2**, BM 170744
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(reduction of risk factors for cardiovascular complications by BM 17.0744)

RN 221564-97-2 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 18 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:759530 HCAPLUS

DOCUMENT NUMBER: 132:73476

TITLE: Species differences in induction of hepatic enzymes by BM 17.0744, an activator of peroxisome proliferator-activated receptor alpha (PPARα)

AUTHOR(S): Meyer, Kirstin; Volkl, Alfred; Endele, Richard; Kuhnle, Hans-Frieder; Pill, Johannes

CORPORATE SOURCE: Therapeutics Research, Roche Diagnostics GmbH, Mannheim, D-68305, Germany

SOURCE: Archives of Toxicology (1999), 73(8-9), 440-450
CODEN: ARTODN; ISSN: 0340-5761

PUBLISHER: Springer-Verlag

DOCUMENT TYPE: Journal

LANGUAGE: English

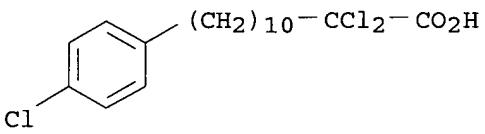
AB BM 17.0744, a new anti-diabetic and lipid-lowering agent, leads also to strong hepatomegaly and carnitine acetyl transferase (CAT) increase in the liver of rats, a phenomenon known from fibrates. For information on the relevance of changes in liver of rats to other species, we investigated the effects of BM 17.0744 on lipids and selected marker enzymes related to β-oxidation in rats, dogs and guinea-pigs, so-called high and low responders to peroxisome proliferators. To examine selectivity other enzymes were also determined, e.g. esterase, urate oxidase (UOX) and cytochrome c oxidase (CYT.C.OX.). Lowering of triglycerides and cholesterol in blood serum and/or liver was observed in pharmacol. dose range in the three species tested. In dogs and guinea-pigs, liver and kidney wts. were unaffected even in dogs in medium and high dose groups with high systemic exposure and severe toxicity. In male Sprague-Dawley rats treatment with 1.5, 3, 6 and 12.5 mg/kg per day BM 17.0744 selectively elevated the activities of CAT and acyl-CoA oxidase (AOX) by ≤200 and 20-fold, resp. Administration of BM 17.0744 to Beagle dogs (1.5, 4, 12 mg/kg per day) and guinea-pigs (3 and 12 mg/kg per day) enhanced the activities of CAT and AOX dose-dependently by a factor of two to three only. Immunoblotting revealed a drug-specific enhancement of the amount of β-oxidation enzymes in rats, which is in accord with the rapid and coordinated transcriptional activation shown in Northern dot blot anal. Nuclear run-on assays

demonstrated a real transcriptional activation. BM 17.0744 activates peroxisome proliferator-activated receptor α (PPARα), which could be shown by transactivation assays. The stimulation of PPARα by BM 17.0744 was stronger than that of the known ligands WY 14.643 and ETYA. Activation of PPARγ can be excluded. Taken collectively, the data demonstrate an enhancement of the β-oxidation system by BM 17.0744 paralleled by lipid-lowering in all species investigated. The activation of the nuclear factor PPARα may explain the changes in liver and the metabolic effects on the mol. level. The lack of an increase in liver and kidney wts. and the relatively moderate enhancement of activities of β-oxidation-related enzymes in dogs and guinea-pigs indicate that the excessive response observed in rats is not applicable to other, predominantly non-rodent, species. On the basis of these data and the experience with fibrates a specific risk for humans is not expected.

IT **221564-97-2**, BM 170744
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(species differences in hepatic enzyme induction by PPARα activator BM 170744)

RN 221564-97-2 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

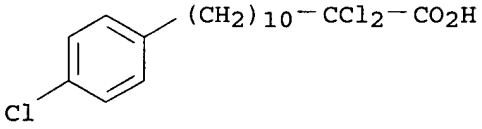
L6 ANSWER 19 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:36671 HCAPLUS
DOCUMENT NUMBER: 130:246780
TITLE: BM 17.0744: a structurally new antidiabetic compound with insulin-sensitizing and lipid-lowering activity
AUTHOR(S): Pill, Johannes; Kuhnle, Hans-Frieder
CORPORATE SOURCE: Therapeutics Research, Boehringer Mannheim, Mannheim, 68305, Germany
SOURCE: Metabolism, Clinical and Experimental (1999), 48(1), 34-40
CODEN: METAAJ; ISSN: 0026-0495
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB BM 17.0744 (2,2-dichloro-12-(p-chlorophenyl)-dodecanoic acid) is a substance from a group of ω-substituted alkyl carboxylic acids with the general formula, ring-spacer-carboxylic acid. With BM 17.0744, a compound structurally unrelated to thiazolidinediones, antihyperglycemic and antihyperinsulinemic potency has been demonstrated in various animal models of type II diabetes. The antidiabetic effect is independent of the genetic background of the disease, gender, and animal species. The 24-h blood glucose profile was dose- and time-dependently improved in ob/ob mice after a single and fourth oral administration of 0.3, 1, and 3 mg/kg/d. A dose-dependent reduction of hyperglycemia (10%, 15%, 28%, and 66%) was found in db/db mice after the fifth oral administration of 3, 10, 30,

and 100 mg/kg/d. Hyperinsulinemia was reduced dose-dependently in yellow KK mice by 1%, 24%, 34%, and 66% after the fifth oral administration of 0.3, 1, 3, and 10 mg/kg/d. Overall glucose metabolism was predominantly higher in euglycemic-hyperinsulinemic clamp studies in obese fa/fa rats pretreated for 14 days with 10 mg/kg/d BM 17.0744. The data in diabetic and insulin-resistant animals suggest an improvement of insulin action that is supported by enhancement of insulin effects in vitro. There is no evidence of a risk for hypoglycemia in diabetic and metabolically healthy animals. Triglyceride (TG) and cholesterol were reduced in the serum of metabolically healthy rats, as well as serum lipids in db/db mice, which suggests this effect is independent of amelioration of the diabetic status. Lipid-lowering effects in diabetic and healthy animals show an addnl. property of BM 17.0744. Because of its antidiabetic and lipid-lowering potency, the substance is of great interest in treating the metabolic syndrome. Lipid decreases in rats are associated with a dose-dependent increase in carnitine acetyltransferase activity in the liver to about 100-fold (12.5 mg/kg/d). This together with hepatomegaly in small rodents may indicate peroxisomal proliferation, a phenomenon considered species-specific. Its relevance for humans is well documented for other classes of compds. including fibrates. Specific side effects of insulin sensitizers of the thiazolidinedione type, such as an increase in body weight and heart weight, could not be observed after 4-wk oral application of BM 17.0744 in rats. In general, BM 17.0744 was well tolerated in the pharmacol. dose range in all species tested.

IT **221564-97-2**, BM 170744
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(BM 17.0744 is antidiabetic compound with insulin-sensitizing and lipid-lowering activity)

RN 221564-97-2 HCAPLUS
CN Benzenedodecanoic acid, α,α,4-trichloro- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 20 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:793822 HCAPLUS
DOCUMENT NUMBER: 130:168061
TITLE: ω-Substituted alkyl carboxylic acids as antidiabetic and lipid-lowering agents
AUTHOR(S): Meyer, Kirstin; Voss, Edgar; Neidlein, Richard; Kuhnle, Hans-Frieder; Pill, Johannes
CORPORATE SOURCE: Therapeutics Research, Boehringer Mannheim, Mannheim, Germany
SOURCE: European Journal of Medicinal Chemistry (1998), 33(10), 775-787
CODEN: EJMCA5; ISSN: 0223-5234
PUBLISHER: Editions Scientifiques et Medicales Elsevier
DOCUMENT TYPE: Journal

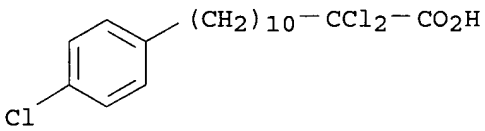
LANGUAGE: English

AB In screening expts. certain ω-substituted alkylcarboxylic acids were found to produce an increase in insulin-stimulated 14C-acetate incorporation into triglycerides, which may indicate an improvement in the action of insulin. Antidiabetic and lipid-lowering properties in genetically diabetic ob/ob mice demonstrated the in vivo relevance of the insulin-potentiating effects seen in vitro. The chemical structures of the ω-substituted alkylcarboxylic acids with insulin-potentiating effects correspond to the general formula ring-spacer-COOH. A close structure-activity relationship was observed The most potent compound in ob/ob mice was 4-ClC6H4(CH2)10CCl2CO2H, which normalized blood glucose as well as hyperinsulinemia and lowered serum triglycerides and cholesterol by 52% and 37%, resp. On the basis of these results, ω-substituted alkylcarboxylic acids are interesting as a new class of oral antidiabetic agents with insulin-sensitizing and lipid-lowering activity.

IT **178671-17-5P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(preparation of aryldichloroalkanoic acids as antidiabetic and hypolipemic agents)

RN 178671-17-5 HCAPLUS

CN Benzenedodecanoic acid, α,α,4-trichloro-, sodium salt (9CI) (CA INDEX NAME)



REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 21 OF 21 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1996:431377 HCAPLUS

DOCUMENT NUMBER: 125:86196

TITLE: Preparation of 2,2-dichloroalkanoic acids as antidiabetic agents

INVENTOR(S): Voss, Edgar; Pill, Johannes; Freund, Peter

PATENT ASSIGNEE(S): Boehringer Mannheim Gmbh, Germany

SOURCE: Ger. Offen., 14 pp.
CODEN: GWXXBX

DOCUMENT TYPE: Patent

LANGUAGE: German

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 4439947	A1	19960515	DE 1994-4439947	19941109
ZA 9509451	A	19970508	ZA 1995-9451	19951108
IL 115920	A1	20031123	IL 1995-115920	19951108
CA 2204527	AA	19960530	CA 1995-2204527	19951109

WO 9615784	A2	19960530	WO 1995-EP4413		19951109
WO 9615784	A3	19960530			
W: AU, BG, BR, BY, CA, CN, CZ, EE, FI, HU, JP, KR, KZ, MX, NO, NZ, PL, RO, RU, SI, SK, UA, US					
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
AU 9641153	A1	19960617	AU 1996-41153		19951109
AU 699480	B2	19981203			
EP 790824	A1	19970827	EP 1995-939248		19951109
EP 790824	B1	20020206			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE					
CN 1171050	A	19980121	CN 1995-197101		19951109
CN 1105558	B	20030416			
HU 77627	A2	19980629	HU 1998-381		19951109
JP 3012004	B2	20000221	JP 1996-516511		19951109
JP 10510515	T2	19981013			
PL 181689	B1	20010928	PL 1995-320167		19951109
AT 212834	E	20020215	AT 1995-939248		19951109
PT 790824	T	20020731	PT 1995-939248		19951109
ES 2170813	T3	20020816	ES 1995-939248		19951109
RU 2197960	C2	20030210	RU 1997-109840		19951109
SK 284019	B6	20040803	SK 1997-568		19951109
CZ 294254	B6	20041110	CZ 1997-1320		19951109
FI 9701951	A	19970707	FI 1997-1951		19970507
NO 9702128	A	19970709	NO 1997-2128		19970507
US 5968982	A	19991019	US 1997-817925		19970708
PRIORITY APPLN. INFO.:			DE 1994-4439947	A	19941109
			WO 1995-EP4413	W	19951109

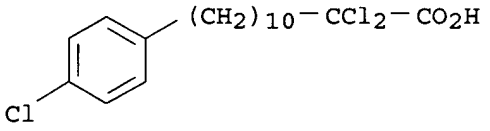
OTHER SOURCE(S): MARPAT 125:86196

AB RZ1Z2Z3CCl2CO2H [R = halo, cyano, Me, cycloalkyl, Ph, etc.; Z1 = bond, CH:CH, C.tplbond.C, alkylene, etc.; Z2 = O, S, (un)substituted imino; Z3 = C5-20 alkylene] were prepared as antidiabetic agents (no data). Thus, Cl2CHCO2H was alkylated by Br(CH2)10Br to give Br(CH2)10CCl2CO2H.

IT **178671-17-5P 178671-34-6P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of 2,2-dichloroalkanoic acids as antidiabetic agents)

RN 178671-17-5 HCAPLUS

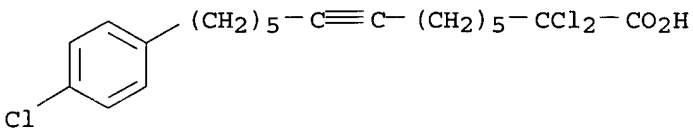
CN Benzenedodecanoic acid, α,α,4-trichloro-, sodium salt (9CI) (CA INDEX NAME)



● Na

RN 178671-34-6 HCAPLUS

CN 8-Tetradecynoic acid, 2,2-dichloro-14-(4-chlorophenyl)- (9CI) (CA INDEX NAME)


(CH2)5—C≡C—(CH2)5—CCl2—CO2H
Cl

=>